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HYPOBARIC DECOMPRESSION SICKNESS MODEL DEVELOPMENT (PART I): DIFFUSION OF INERT GAS FROM A VISCOELASTIC FLUID (BLOOD) INTO AN EXPANDING GAS PHASE

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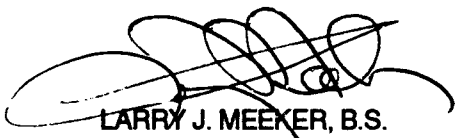

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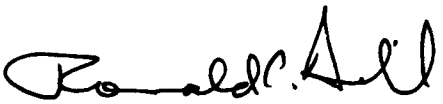

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Bubble growth within a volume of isothermal viscoelastic liquid containing uniformly distributed dissolved gas is considered. A nonlinear viscoelastic constitutive equation is used as a blood model. The problem of characterizing this growth-by-mass-transfer is being treated extensively, in order to better understand both the behavior of bubble growth due to supersaturation and its effects on altitude decompression sickness.

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Hypobaric Decompression Sickness Model Development (Part I): Diffusion of Inert Gas from a Viscoelastic Fluid (Blood) into an Expanding Gas Phase

Introduction

In 1989, an altitude decompression model development program was initiated at the Armstrong Laboratory. The immediate goal of this effort is to define the architecture (or framework) and the algorithms (or software) for the decompression model. The final product will be a software package that will serve as the altitude decompression "standard" and will lead to hardware development to provide the USAF with DCS risk assessment capability for a variety of operational settings. This report is one in a series of several steps taken towards the completion of this goal.

In studies on cavitation and cavitation damage, it is necessary to use mathematical expressions to characterize pressure and velocity fields in the neighborhood of a growing or collapsing gas-filled cavity in a fluid. Our emphasis is on the problem of spherical bubbles in an incompressible viscoelastic liquid. Attempts have been made recently to associate the number of bubbles and their size, with the existence and severity of decompression sickness (DCS). It is a fact that bubbles appear when dissolved gases are supersaturated in the blood and the tissue during DCS. Bubbles have been seen with various methods (Ultrasound imaging techniques), and it is commonly believed that their size and numbers are involved in DCS. The direct relationship is yet to be discovered because it is still not known how to relate critical variables (that will be able to pre-inform us if and when DCS will occur and where) with the actual physiological processes. The details of the processes underlying some of the manifestations of altitude decompression sickness remain unknown, but the basic mechanism is undoubtedly the supersaturation of the tissues and blood with nitrogen. DCS may arise either in flight or during exposure to reduced atmospheric pressure in a hypobaric chamber. The dynamic representation of the diffusion process, and the understanding of its solutions, analytical (if analytical solutions are possible) or numerical, will provide an important tool to better understand this complex physiological system.

Effects of viscoelasticity and compressibility on the bubble dynamics have been previously investigated. Barlow and Langlois[1] considered the diffusion-fed growth of a spherical gas bubble into a Newtonian viscous liquid under isothermal conditions. Street[6] performed a theoretical study of bubble growth in a viscoelastic medium (represented by the Oldroyd three-constant model) by considering the momentum transfer process, but neglected a diffusion process between the gas and the liquid phase. Tanasawa and Yang[7] also investigated the collapse of a gas bubble suspended in a viscoelastic liquid represented by the Oldroyd three-constant model. Like Street, they neglected the diffusion process between the gas and the liquid phase because they made the assumption that the gas inside the bubble was undergoing a reversible polytropic process (the instantaneous gas pressure inside the bubble was a function of a polytropic exponent and not coupled with a diffusion process). By taking into account the thermodynamic behavior of the gas phase inside the bubble, they observed that, when the bubble oscillation occurs during collapse, viscous damping is less important in viscoelastic liquids than it is in purely viscous liquids. They observed further that a gas bubble collapses faster under adiabatic conditions than under isothermal conditions. We will investigate a similar case, but with the exception that we will take under consideration the fact that the blood diffusion rate cannot be characterized either fast nor slow (for this reason the diffusion equation cannot be neglected). If the diffusion coefficient of a gas dissolved in the suspending medium lies in the intermediate range, we must solve the hydrodynamic and the diffusion equation simultaneously.

Assumptions

We assume, first of all, that a single bubble of gas is growing in an otherwise unlimited volume of liquid in the absence of foam dynamics (in other words we assume that bubbles won't compete for space with others growing in close proximity). We assume that the liquid is a viscoelastic fluid, and we will use an Oldroyd type B model to represent the rheological properties of the blood. We also assume that the liquid is incompressible (low velocity gas flow can be assumed to be incompressible); the gas is ideal, it diffuses according to Fick's law. The concentration inside the bubble is related to the concentration just outside the bubble according to Henry's law, and only a negligible amount of gas is absorbed on the bubble wall. Once we reach the desirable altitude, the initial radius value of the gas pocket will change according to Boyle's law (This change occurs once and is discrete). The Laplace relationship is valid because we emphasize growth and not nucleation. The bubbles are assumed to be spherical.

Hydrodynamics of the Model

First we must define the hydrodynamics of the problem. When a bubble grows in a liquid, there will be a velocity field within the liquid at the gas-liquid interface, which in turn, generates a stress field tending to retard the bubble's growth. Basically, when the fluid is being pushed by the growing bubble, it creates a velocity field, which is equal to the rate of change of the bubble radius. The spherical symmetry of the situation (the bubbles are spherical), makes it convenient to choose a spherical coordinate system with its origin at the bubble center. The relationship of our hydrodynamic equation and the diffusion equation is governed by the diffusion rate. Peticolas[3] calculated the limiting cases of extremely rapid and extremely slow gas diffusion, and he came to the following conclusion: if the diffusion is sufficiently rapid, the bubble pressure remains constant and the growth is determined by the hydrodynamic equations alone. Here the treatment of the moving boundary is important. On the other hand, if the diffusion is slow, hydrodynamic effects become negligible, and the diffusion equation alone describes the process fairly accurately. An asymptotic solution of the second case, for viscous liquids, was derived by Barlow and Langlois[1] in 1962. We will solve the intermediate case where the hydrodynamic equation is coupled with the diffusion equation.

The Model

The velocity field generated in the liquid can be described completely by the Navier-Stokes equations. If we assume that it has only a radial component v(r,t), it reduces to: $v_r=v_r(r,t)$, $v_\theta=v_\varphi=0$. From the conservation of momentum equation:

$$\rho\left(\frac{\partial V_r}{\partial t}+V_r\frac{\partial V_r}{\partial r}\right)=-\frac{\partial p}{\partial r}+\frac{1}{r^2}\frac{\partial}{\partial r}(r^2\tau_m)-\frac{(\tau_{\theta\theta}+\tau_{\varphi\varphi})}{r} \qquad (1)$$

From the continuity equation in spherical coordinates (See appendix 1):

$$\frac{\partial}{\partial r}(r^2V_r)=0 \qquad (2)$$

Integrating equation (2) from R(t) to r we get:

$$V_r=\frac{R^2\dot{R}}{r^2} \qquad (3)$$

Notice that we treat a stationary bubble, with respect to the blood flow (creeping flow is considered in the veins). We can assume, because of the biomolecular structure of the fluid, that the bubble (like a suspension) is just hitching a ride with the blood. Once it is nucleated, it doesn't move independently. It just follows the flow (Buoyancy forces, gravity effect are omitted), and it grows in an infinite mass of homogeneous incompressible viscoelastic liquid. Incompressibility and spherical symmetry implies $\tau_{rr}+\tau_{\theta\theta}+\tau_{\varphi\varphi}=0$ and $\tau_{\theta\theta}=\tau_{\varphi\varphi}$. By taking under consideration these two relationships and by substituting equation 2 and 3 in equation 1, we integrate equation 1 (with respect to r) from R to infinity. The result is the following integrodifferential equation:

$$\rho\left[\frac{\ddot{R}R^2}{r}+\dot{R}^2\left(\frac{2R}{r}-\frac{R^4}{2r^4}\right)\right]_{r=R}=p_f(R)-p(\infty)+\tau_{rr}(\infty)-\tau_{rr,f}(R)+3\int_r^{\infty}\frac{\tau_{rr}}{r}dr \qquad (4)$$

The above equation represents the pressure distribution in the blood. The dynamic equation governing the growth or collapse of the bubble may be obtained by replacing r with R. The balance of forces at the bubble surface (r=R) requires that:

$$p_f(R)=p_g(R)-\left(\frac{2\sigma}{R}\right)+\tau_{rr,f}(R) \qquad (5)$$

in which the radial normal stress acting on the bubble surface due to the gas phase viscocity is neglected. Similarly, the balance of forces at r=∞ will be: $-p(\infty)+\tau_{rr}(\infty)=-p_\infty$ (Relationship 1).

In the present study, the three-parameter, Oldroyd model is employed to represent the rheological behavior of the blood.[4] The B fluid model is proposed here as the simplest case that might apply to blood (shear thinning, positive Weissenberg effect). This model allows a reduction to a three-constant model but results in both the elimination of the extra normal stress terms and the modification of the term $\tau_{\theta\theta}$ in a viscometric flow. However, since it does not appear that one can determine the additional constants experimentally, the model is employed as a reasonable approach in the spirit of most models proposed for the complex blood fluid. Other more recent models for the blood are the three-dimensional dyadic Walburn-Schneck constitutive equation derived by Easthope[2] and the non-linear Maxwell model by Quemada[12]. Both of these models will be examined in the future.

The full constitutive equation for the blood was given by Phillips and Deutsch in their 1975 paper.[4] Here we will treat the three-parameter model, which can be written in spherical coordinates:

$$\tau_{ik}+\lambda_1\left(\frac{D\tau_{ik}}{Dt}\right)=2\eta_d\left[d_{ik}+\lambda_2\left(\frac{Dd_{ik}}{Dt}\right)\right] \qquad (6)$$

where D/Dt is the material derivative defined:

$$\frac{D}{Dt}=\frac{\partial}{\partial t}+(V\cdot\nabla) \qquad (7)$$

τ_{ik} means that the stress variable is a tensor, and it is in a matrix form. Because of our assumptions, τ is equal to the following matrix:

$$\tau_{ik}=\begin{pmatrix}\tau_{rr} & 0 & 0\\ 0 & \tau_{\theta\theta} & 0\\ 0 & 0 & \tau_{\varphi\varphi}\end{pmatrix} \tag{8}$$

λ_1 is the characteristic stress-relaxation time, η_o is the shear viscosity, λ_2 is the characteristic strain-relaxation time ($\lambda_1 \geq \lambda_2 \geq 0$), and d_{ik} is the rate of strain tensor which can be expressed:

$$d_{ik}=\begin{pmatrix}d_{rr} & 0 & 0\\ 0 & d_{\theta\theta} & 0\\ 0 & 0 & d_{\varphi\varphi}\end{pmatrix}=\begin{pmatrix}\frac{\partial V}{\partial r} & 0 & 0\\ 0 & \frac{V}{r} & 0\\ 0 & 0 & \frac{V}{r}\end{pmatrix} \tag{9}$$

The special case in which $\lambda_1=\lambda_2=0$ corresponds to a Newtonian fluid. Remember equation 6 is in matrix form. The stress tensor is being represented by equation 8; consequently, equation 6 is actually a system of 3 differential equations. Because we are talking about a velocity field in the liquid, we are interested only in the radial stress component. Thus, the matrix form of equation 6 can be reduced to the following:

$$\tau_{rr}+\lambda_1\left(\frac{D\tau_{rr}}{Dt}\right)=2\eta_o\left[\frac{\partial V}{\partial r}+\lambda_2\frac{D}{Dt}\left(\frac{\partial V}{\partial r}\right)\right] \tag{10}$$

We use the same technique that Tanasawa and Yang used in their 1970 paper[7], in order to avoid nonlinear convective terms by introducing a new independent variable: $y=(r^3-[R(t)]^3)/3$. The material derivative defined in equation 7 in the (r,t) coordinate system may be reduced to the following form in the (y,t) coordinate system:

$$\frac{D}{Dt}=\frac{\partial}{\partial t}-R^2\dot{R}\frac{\partial}{\partial y}+\frac{R^2\dot{R}}{r^2}r^2\frac{\partial}{\partial y}=\frac{\partial}{\partial t} \tag{11}$$

Therefore equation 10 can be written:

$$\lambda_1\left[\frac{\partial\tau_{rr}(y,t)}{\partial t}\right]+\tau_{rr}(y,t)=-4\eta_o(Z+\lambda_2\dot{Z}) \tag{12}$$

Where $\dot{Z}=\partial Z/\partial t$ and

$$Z=\frac{R^2\dot{R}}{(3y+R^3)} \tag{13}$$

Thus, through this transformation, the non-linear convective terms in equation 10 are eliminated. The normal stress component can be obtained by integrating equation 12 as:

$$\tau_{rr}(y,t) = -\frac{4\eta_o}{\lambda_1}\int_0^t \exp\left(\frac{\xi - t}{\lambda_1}\right)\frac{R^2\dot{R} + \lambda_2(R^2\ddot{R} + 2R\dot{R}^2)}{3y + R^3}\,d\xi \tag{14}$$

Finally equations 4,5,14 and relationship 1 are combined and the resulting equation is integrated with respect to y. It yields:

$$\rho\left(R\ddot{R} + \frac{3}{2}\dot{R}^2\right) = p_g(t)_{r=R} - p_\infty - \frac{2\sigma}{R} \tag{15}$$

$$-\frac{12\eta_o}{\lambda_1}\int_0^t \exp\left(\frac{\xi - t}{\lambda_1}\right)\frac{R^2(\xi)\dot{R}(\xi) + \lambda_2[R^2(\xi)\ddot{R}(\xi) + 2R(\xi)\dot{R}^2(\xi)]}{R^3(t) - R^3(\xi)}\ln\frac{R(t)}{R(\xi)}\,d\xi$$

This relationship is an integro-differential equation that tells us how the bubble radius varies with respect to time. In addition to R, we have another variable whose rate we also have to define. The pressure p_l at any point in the liquid is a function of r and t. However the pressure p_g (pressure of the ideal gas forming the bubble) is only a function of time. We need an expression that relates P_g to bubble radius R. Such a relationship can be derived from a mass balance performed on the gas dissolved in the liquid phase by Fick's law of diffusion. Before we proceed in describing the process, it should be mentioned that the bubble pressure $p_g(t)$ is related to the amount of gas within the bubble at time t according to the ideal gas law. If we assume isothermal conditions, (in our case this assumption is perfectly valid, because we keep the cockpit at constant temperature): $p_g(t)=A\rho_g(t)$(Relationship 2), where ρ_g is the concentration (density) of the gas forming the bubble, and where A is a constant given by $A=R_gT/M$. Constant R_g is the universal gas constant, T is the absolute temperature, and M is the molecular weight of the gas (nitrogen in our case).

Assume that at time t=0, a homogeneous concentration C_o of gas is dissolved throughout the liquid. As mentioned above, the concentration C obeys Fick's law of diffusion, and in spherical coordinates has the form:

$$\frac{\partial C_l}{\partial t} + V_r\frac{\partial C_l}{\partial r} = D\left[\frac{1}{r^2}\frac{\partial}{\partial r}\left(r^2\frac{\partial C_l}{\partial r}\right)\right] \tag{16}$$

where $V_r=R^2(dR/dt)/r^2$ and D is the diffusion constant. The material derivative is used to account for convection of gas by the moving liquid. At the time the pilot first reaches altitude the bubble (actually a nuclei, previously in equilibrium at sea level conditions, being created through heterogeneous nucleation or tribonucleation) will grow instantly (Boyle's law), resulting in a discrete drop in pressure inside the bubble. As the gas bubble grows, the pressure (concentration) inside the bubble will decrease, giving rise to a [1]concentration gradient at the gas-liquid interface, which in turn, affects the mass transfer process (This concentration gradient depends on the altitude that the pilot was exposed to, and the prebreathe†

[1]† prebreathe=100% Oxygen intake prior to exposure to maximum altitude.

protocol). Hence equation 16 must be solved simultaneously with an expression that relates the solute concentration just outside the bubble wall, $C_w(t)$, to the concentration inside the gas bubble, $C_g(t)$. Henry's law will provide that relationship for us: $C_l(R,t)-C_w(t)=K_pP_g(t)=K_pA\rho_g(t)$. Then $\rho_g(t)=C_w(t)/K_pA$ (Relationship 3). Constant K_p is the Henry's law constant. The initial conditions for R(t) and $C_l(r,t)$ may be written as $R(0)=R_o$, $dR(0)/dt=0$, and $C_l(r,0)=C_o$.

When the arterial nitrogen concentration drops, either because of altitude exposure or because of an increase of the oxygen concentration in the breathing air, the venule nitrogen partial pressure depends on the capillary diffusion-perfusion gas exchange process between the blood and the tissue. This exchange process serves as a control mechanism of the venule nitrogen partial pressure. An increase in the venule nitrogen partial pressure results in a concentration gradient between the blood and the bubble, and the activation of the growth process. For this reason, our analysis is better applied in bubbles that nucleate and grow in the veins. Here, the nitrogen concentration does not vary greatly with distance; that's why we will assume that, at a relative distance from the bubble, the concentration of nitrogen will be constant. (In general this is not true. In order to understand how the nitrogen concentration changes axially, radially and due to convection in the veins, we must fully solve and understand the parabolic transport model, describing capillary gas exchange properties. This has been the topic of several papers by Goresky[13], and Bassingthwaighte[14]). Translating this assumption into mathematics provides us with our first boundary condition:

$$\lim_{r\to\infty} C_l(r,t)=C_o \tag{17}$$

At a sufficiently great distance from the gas-liquid interface, the effect of the growing bubble should be negligible; thus, the concentration at any time is nearly equal to the initial concentration. The second boundary condition representing the mass flux at the boundary can be expressed:

$$\frac{d(R^3\rho_g)}{dt}=3\rho_l R^2 D\left(\frac{\partial C_l}{\partial r}\right)\Big|_{r=R} \tag{18}$$

In order to gain information on bubble growth rate, we must solve equations 15, 16 and 18 simultaneously. In so doing, we shall adopt the integral or moment method (one member of a class of so-called "weighted residual" methods) introduced by Rosner and Epstein[5], which permits us to obtain an expression for the bubble radius-time relationship in closed form. It provides a practical and sufficiently accurate approach to these nonlinear, multiparameter boundary value problems. As Rosner and Epstein mentioned in their paper, this method is particularly well suited to problems in which the dependent variable (solute concentration in our case) at the liquid-vapor interface is time-dependent. Applying this method, we manage to reduce our partial differential system to a system of ordinary differential equations in which time is the (only) independent variable.

Multiplying both sides of equation 16 by r^2 and integrating the resulting equation with respect to r from r=R to r=R+δ yields:

$$\int_R^{R+\delta} r^2\frac{\partial C_l}{\partial t}dr+\int_R^{R+\delta} r^2 V_r\frac{\partial C_l}{\partial r}dr=D\int_R^{R+\delta} d\left(r^2\frac{\partial C_l}{\partial r}\right) \tag{19}$$

in which δ is a thin concentration boundary layer. Next we integrate each term taking under consideration the following relationships: $r^2V_r=R^2R$, $C_l(R+\delta)=C_o$, $C_l(R)=C_w$, $\partial C_l/\partial r=0$ at $r=R+\delta$. Thus:

$$\frac{d}{dt}\int_R^{R+\delta}(C_l-C_o)r^2dr=-DR^2\left(\frac{\partial C_l}{\partial r}\right)_{r=R} \tag{20}$$

where C_o is the concentration of the gas dissolved in the blood far from the bubble, which as we mentioned previously, may be considered to remain constant during the entire period of bubble growth. Combining equations 18 and 20, and integrating the resulting equation with respect to time:

$$3\rho_l\int_R^{R+\delta}\left(\frac{C_o-C_l}{C_o-C_w}\right)r^2dr=\frac{\rho_gR^3-\rho_{go}R_o^3}{C_o-C_w} \tag{21}$$

where ρ_{go} is the density of the gas bubble at t=0, and R_o is the initial bubble radius. Now consider the following class of profiles for the normalized solute concentration defect:

$$\frac{C_\infty-C_l}{C_\infty-C_w}=\begin{bmatrix}(1-\zeta)^2 \text{ for } R\le r\le R+\delta\\ 0 \text{ for } r>R+\delta\end{bmatrix} \tag{22}$$

in which $C_\infty=C_o$ because of boundary conditions, and $\zeta=(r-R)/\delta$. Substitution of equation 22 into the left-hand-side of 21 yields:

$$3\rho\left[\frac{1}{30}\left(\frac{\delta}{R}\right)^3+\frac{1}{6}\left(\frac{\delta}{R}\right)^2+\frac{1}{3}\left(\frac{\delta}{R}\right)\right]R^3=\frac{\rho_gR^3-\rho_{go}R_o^3}{C_o-C_w} \tag{23}$$

We assume that $\delta/R\ll1$; thus, we can disregard the terms $(\delta/R)^2$ and $(\delta/R)^3$ in equation 23 because they are very small. Then, equation 23 becomes:

$$\delta=\frac{\rho_gR^3-\rho_{go}R_o^3}{\rho_l(C_o-C_w)R^2} \tag{24}$$

Substituting equation 22 into the right hand of equation 18 and eliminating δ from the resulting expression, with the aid of equation 24:

$$\frac{d}{dt}(\rho_gR^3)=\frac{6\rho_l^2D(C_o-C_w)^2R^4}{\rho_gR^3-\rho_{go}R_o^3} \tag{25}$$

Equations 15 and 25 must be solved simultaneously for R(t) and $C_w(t)$ with the aid of relationships 2 and 3, using the following initial conditions: $R(0)=R_o$, $dR(0)/dt=0$, $C_w(0)=C_o$.

Discussion

We have developed a bubble growth model for the blood. Special attention must be paid to two of the initial values involved in our computations. The first one is the dR(0)/dt value. We assumed that this value was equal to zero, but this assumption was not totally valid. If we look into the nucleation process that takes place before growth, the size of the embryo (nucleus) constantly changes until it reaches a critical radius size. Embryos that never reach this size soon collapse. The ones that do manage to reach this r-critical radius size (through energy fluctuations), are destined to grow. In our analysis, we "peak-up" the growth process exactly when the embryo crosses the r-critical borderline and becomes a bubble. This threshold means that the rate of radius growth at t=0 (which is the starting time for the growth process, but not for the nucleation process) is not actually zero. However, there is a case in which the initial radius rate of change might, in fact, be zero. There are theories that claim the pre-existence of nuclei in stable form In either case, the variable initial radius growth rates do not affect our final radius computations. Barlow et al.[1] solved analytically the initial stage of growth, at a low Raynolds number, for the viscous case, and he concluded the following:" Except for the first moments of growth, the solution is virtually independent of the value chosen for dR(0)/dt". Thus, none of the two above assumptions will produce a significant error in our calculations. Nevertheless, in this study, we will assume that dR(0)/dt is equal to zero.

The second initial value that we have to pay extra attention to is the initial nitrogen concentration in the blood (on the venous side). This issue is slightly more complicated than the previous one. Prebreathing oxygen before or during the flight (and/or exposure to altitude) affects the pilot's blood and tissue nitrogen concentration levels. All body tissues in equilibrium with ambient nitrogen will respond to increased or decreased nitrogen partial pressure in the breathing medium by absorbing or eliminating dissolved gas in the tissues. The nitrogen partial pressure of the blood in the arterial side can decrease very rapidly; in the case of 100% O_2 prebreathe, it can drop to zero, creating a large concentration gradient between the tissue and the blood in the capillary level. Consequently, the blood begins to wash out nitrogen from the tissue through diffusion. The tissue can be characterized by a spectrum of theoretical tissue types that represent "fast" and "slow" tissues. The outgoing nitrogen partial pressure in the blood (outgoing meaning from the capillary level to the venous side, basically the venous nitrogen partial pressure), can vary depending on the "half-time" of the tissue. A theoretical tissue type is characterized by the tissue half-time: the time required for a tissue to respond to a change in ambient nitrogen partial pressure by giving off or absorbing nitrogen until the initial difference between the tissue nitrogen partial pressure and ambient nitrogen partial pressure is reduced by one-half. Half-time tissues are very helpful if one follows the exponential function approach. (Since we haven't developed our own parabolic transport model yet, we will use the exponential function approach to calculate the nitrogen concentration near the venule walls.)

The outgoing nitrogen partial pressure must be quantified before we can resume our computations. The nitrogen gas dynamics and exchange in the capillaries is not a simple mathematical interpretation. For the purpose of this report, we will compute bubble growth at the venule walls because that's where the nitrogen concentration is at its maximum (maximum concentration in the blood results in a maximum concentration gradient between the blood and the bubble). Its value will be calculated by the use of the washout exponential model developed by Conkin et al.[8] This method is used because, even if we consider the perfusion-diffusion process, the boundary condition in the capillaries will be $C(r=a,t)=C_t \approx C_v$ (a=the radius value of the capillary; C_t is the tissue nitrogen concentration, near the capillary wall; C_v is the venule nitrogen concentration near the vessel's wall and close to the venule-capillary interface). C_t will vary according to the Conkin et al. equation and so will the nitrogen concentration in the venule walls (We are expecting the nitrogen concentration value to be higher in the venule walls no matter what kind of analysis we use, because of the radial diffusion process in the capillaries.). It is logical (for the above reason) to expect most bubbles to form closer to the walls.

Thus, the tissue, depending on its half-time value, alters its partial pressure value due to the concentration gradient between the tissue and the blood. This value can be calculated through the standard Haldanian approach from the following equation:

$$P_t = P_o + (P_a - P_o)(1 - e^{-kt}) \tag{26}$$

where P_t is the nitrogen partial pressure in the tissue after exposure for t minutes; P_o is the initial tissue nitrogen partial pressure; P_a is the ambient nitrogen partial pressure in the breathing medium; t is the exposure time in minutes; and k is the tissue nitrogen partial pressure rate constant ($k=.693/t_{1/2}$ where $t_{1/2}$ is the tissue nitrogen partial pressure half time). Now we can solve our model taking under consideration an array of half-time tissues.

Clarifications

1. It is customary to express the gas concentration in a tissue in terms of its partial pressure p and the solubility α of that gas in the tissue: $C \equiv \alpha P$. In this report, concentration and partial pressure are used constantly; hence, it was necessary to mention their above relationship to avoid confusion. Also we assume that concentration changes in the blood (in the veins, due to convection) are much slower than bubble growth rates.

2. There was no particular reason why we chose an exponential decay approach (a perfusion dominated approach) rather than Hills'[11] approach (a diffusion dominated approach). Hills' equivalent equation for pure radial diffusion can be expressed as:

$$\frac{dp}{dt} = \frac{2D_c}{a} \frac{S_p}{\left(1 + \frac{1-\alpha^2}{\alpha^2} S_p\right)} \left(\frac{\partial p_c}{\partial r}\right)_{r=a} \tag{27}$$

and the above equation is valid only if v (the capillary blood velocity) >>> l (the capillary length). What each of the parameters and variables represent is not important since it is clear now that the diffusion-perfusion model must be solved in order to thoroughly understand capillary transport phenomena and nitrogen concentration values in the veins. This will be the purpose of the second publication in this series.

3. Using polynomial profiles for the gas concentration is tantamount to assuming that the bubble grows in an infinite pool of liquid. In situations where a large number of bubbles nucleate and grow simultaneously in close proximity, the amount of liquid immediately surrounding a bubble is finite and so is the amount of the dissolved gas. Therefore, the hydrodynamics of the growth and the diffusion process will be different from the case of a bubble growing in an infinite medium. "The underlying assumption in adopting a polynomial profile to describe the gas concentration is that at large distances from the bubble interface, the gas concentration remains unchanged and is equal to the initial concentration. This assumption is not valid for a bubble surrounded by a finite amount of liquid with a limited concentration of dissolved gas and cannot predict the steady-state bubble radius correctly."[9] Therefore, it is necessary to solve the diffusion equation in its complete form numerically as opposed to using similarity solutions[10] or approximate analytical methods. In their paper, Michaelides et al.[9] analyzed the process of mass diffusion-induced growth of a gas bubble surrounded by a limited amount of liquid and dissolved gas and presented a solution technique to accurately solve for the concentration profile of the dissolved gas. Bubble growth dynamics was predicted with this concentration profile. As mentioned in our assumptions, we will not include foam dynamics, so the use of polynomial profiles for the gas concentration is sufficient.

Model Summary

The equations that describe bubble growth in the blood, after all the mathematical manipulations, can be summarized in the following system of ordinary differential equations:

$$\rho\left(R\ddot{R}+\frac{3}{2}\dot{R}^2\right)=P_g(t)_{r=R}-P_\infty-\frac{2\sigma}{R}$$

$$-\frac{12\eta_o}{\lambda_1}\int_0^t \exp\left(\frac{\xi-t}{\lambda_1}\right)\frac{R^2(\xi)\dot{R}(\xi)+\lambda_2[R^2(\xi)\ddot{R}(\xi)+2R(\xi)\dot{R}^2(\xi)]}{R^3(t)-R^3(\xi)}\ln\frac{R(t)}{R(\xi)}d\xi$$

And from the mass transfer process:

$$\frac{d}{dt}(\rho_g R^3)=\frac{6\rho_l^2 D(C_o-C_w)^2R^4}{\rho_g R^3-\rho_{go}R_o^3}$$

and
$P_g(t)=A\rho_g(t)$
$C_w(t)=K_pP_g(t)$
With the following initial conditions: $R(0)=R_o$, $dR(0)/dt=0$, $C_w(0)=C_o$

Notation

R =bubble radius
ρ_l =density of the liquid phase
ρ_g =density of the gas inside the bubble
ρ_{go}=density of the gas bubble at t=0
P_g =pressure inside the gas bubble
P_∞ =pressure of the liquid phase far away from the bubble surface
σ =surface tension
A =ideal gas law constant (for a more detailed definition look at page 5)
K_p =Henry's constant
η_o =shear viscosity
λ_1 =characteristic stress relaxation time
ξ =dummy variable
t =time
λ_2 =characteristic strain relaxation time
D =diffusion coefficient
C_o =initial concentration of the gas dissolved in the liquid phase
C_w =solute concentration just outside the bubble wall

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Appendix 1

The momentum equation in spherical coordinates (r,θ,φ) in terms of τ is expressed as follows in the r component:

$$\rho\left(\frac{\partial v_r}{\partial t}+v_r\frac{\partial v_r}{\partial r}+\frac{v_\theta}{r}\frac{\partial v_r}{\partial \theta}+\frac{v_\varphi}{r\sin\theta}\frac{\partial v_r}{\partial \varphi}-\frac{v_\theta^2+v_\varphi^2}{r}\right)=-\frac{\partial p}{\partial r}$$

$$-\left(\frac{1}{r^2}\frac{\partial}{\partial r}(r^2\tau_{rr})+\frac{1}{r\sin\theta}\frac{\partial}{\partial \theta}(\tau_{r\theta}\sin\theta)+\frac{1}{r\sin\theta}\frac{\partial \tau_{r\varphi}}{\partial \varphi}-\frac{\tau_{\theta\theta}+\tau_{\varphi\varphi}}{r}\right)$$

The velocity field generated in the liquid will have only a radial component v(r,t), where t is time measured from the instant of bubble formation. All the derivatives with respect to θ and φ will be zero. Also the velocity components in the θ and φ coordinates will be zero. Taking under consideration the above assumptions, the momentum equation can be rewritten as follows:

$$\rho\left(\frac{\partial v_r}{\partial t}+v_r\frac{\partial v_r}{\partial r}\right)=-\frac{\partial p}{\partial r}+\frac{1}{r^2}\frac{\partial}{\partial r}(r^2\tau_{rr})-\frac{(\tau_{\theta\theta}+\tau_{\varphi\varphi})}{r}$$

The equation of continuity in spherical coordinates (r,θ,φ) can be expressed as follows:

$$\frac{\partial \rho}{\partial t}+\frac{1}{r^2}\frac{\partial}{\partial r}(\rho r^2 v_r)+\frac{1}{r\sin\theta}\frac{\partial}{\partial \theta}(\rho v_\theta\sin\theta)+\frac{1}{r\sin\theta}\frac{\partial}{\partial \varphi}(\rho v_\varphi)=0$$

As we mentioned before, all partial derivatives with respect to θ and φ, will be zero. The density of the fluid is not a spatial variable; therefore, the above equation can be reduced to the following one:

$$\frac{1}{r^2}\frac{\partial}{\partial r}(\rho r^2 v_r)=0=\frac{\partial}{\partial r}(r^2 v_r)$$